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# A VASCULAR WILT OF THE MIMOSA TREE [1] (ALBIZZIA JULIBRISSIN)

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## INTRODUCTION

The so-called mimosa tree (*Albizzia julibrissin* Duraz.), a native of eastern Asia, is widely grown in the southern part of the United States as an ornamental. It grows very rapidly and has graceful fernlike leaves and striking colorful flowers. In recent years the tree has gained considerably in popularity and is now commonly used along roadsides, because of both its beauty and its capacity to thrive under unfavorable soil conditions.

During the summer of 1935 the attention of the Division of Forest Pathology was called to the rapid dying of mimosa trees in the town of Tryon, N. C. Local residents reported this dying to have begun about 1930 and to have been increasing since. Trees rapidly wilted and never recovered. The situation at Tryon, where several hundred trees were dead and dying, was studied in some detail. As a result of this examination a brief article was prepared [3] and information on the disease was disseminated. No intensive scouting has been done, but trees in localities where the species is common, particularly in the Carolinas, have been under observation by agencies and individuals apprised of the disease. Additional infections have been discovered in or near the following towns: Richmond, Emporia, and Norfolk, Va.; Biltmore, Gastonia, Dallas, Belmont, Mount Holly, Charlotte, Pineville, Polkton, Wadesboro, Lilesville, Morven, Pee Dee, Hillsboro, Raleigh, Knightdale, Durham, and Louisburg, N. C.;

[1] Throughout the South generally called mimosa or mimosa tree; in the literature known as the silktree and silky acacia.

[2] The writer is indebted to H. W. Wollenweber, of the Biological Institute in Berlin-Dahlem, and C. D. Sherbakoff, agent, Division of Cotton and Other Fiber Crops and Diseases, for the identification of fungi and for their counsel during this investigation, and to Edith K. Cash, of the Division of Mycology and Disease Survey, for preparation of the Latin description.

[3] HEPTING, G. H. A DESTRUCTIVE DISEASE OF THE MIMOSA TREE IN THE CAROLINAS. U. S. Bur. Plant Indus. Plant Disease Reptr. 20: 177–178. 1936. [Mimeographed.]

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Spartanburg, Clover, Osceola, Van Wyck, Lancaster, Great Falls, Winnsboro, Society Hill, Wisacky, Parler, Columbia, Johnston, and Edgefield, S. C.; and Monticello and La Grange, Ga. It is probable that the disease also occurs elsewhere in this section. At some of the places only an occasional shade tree has become infected, but at others, particularly where the species has regenerated prolifically along roads, hundreds of trees are dead and dying. No evidence has been found to indicate that trees were diseased prior to 1934 except at Tryon. The estimate of time of appearance of the disease is fairly accurate because most of the diseased trees are growing about habitations and hence have been under constant observation.

## SYMPTOMS

The first external symptom of the disease is wilting of the leaves on some of the branches (figs. 1, *B*, and 2, *A*), causing the foliage to hang downward. The wilted leaves then become dry and shriveled and soon fall. Shortly afterward the foliage on the remaining branches wilts and dies. Death follows defoliation, which usually is complete within a year of the time the first wilting is apparent. However, entire trees, 15 to 20 feet tall, have been found completely wilted within a month from the time wilting was first observed. In some cases where progressive wilting is gradual, dwarfed chlorotic leaves are produced prior to death.

By the time wilting takes place, a brown ring or partial ring of discolored sapwood can be found in the trunk, usually in the current annual ring (fig. 2, *B*). This discoloration extends out the branches, but often it is not evident in the smaller ones until the later stages of the disease. The discoloration was traced throughout its length in several small trees; it appeared heaviest in the roots and became less intensive proceeding up the stem and out the branches. Sections through the discolored sapwood of the trunk showed fungus hyphae in all the xylem elements (fig. 2, *C* and *D*). Sections made through such wood well out on branches where the discoloration was slight showed hyphae in the vessels only. The contents of the xylem elements that had been living when invaded, including ray cells and wood parenchyma, apparently became transformed to a brown gummy substance by some reaction with the fungus (fig. 2, *C* and *E*). Where this substance is produced in parenchyma cells surrounding vessels, it apparently exudes into the vessels, thereby plugging them (fig. 2, *E*). In a cross section of wood in the infected zone, viewed under the microscope, ray and other parenchyma cells stand out sharply because of their dark-brown color.

A disease of *Albizzia julibrissin* having the identical symptomatology and the epidemic nature of this disease was described from the southern Soviet Union in 1920.[4] Characteristics common both to the Russian and the American diseases include: (1) Wilting followed by defoliation and death, (2) the presence of a brown ring of discoloration usually in the last annual ring, (3) the occurrence of a brown substance in the vessels, parenchyma, and ray cells, and (4) the presence of mycelium in the elements of the discolored xylem.

[4] VORONIKHIN, N. N. FUSARIUM ALBIZZIAE NOV. SP., AS A POSSIBLE CAUSE OF THE DESTRUCTION OF THE SILKY ACACIA (ALBIZZIA JULIBRISSIN BOISS.) IN THE BATUM REGION. [Translated title.] Vest. Tiflis. Bot. Sada 48: 15–36. 1920.

FIGURE 1.—*A*, Normal branch of a mimosa tree; *B*, part of a diseased tree with wilted foliage, the first external symptom of the disease.

Two seedling diseases of *Albizzia julibrissin* have been reported from Japan. Both diseases were confined to first-year nursery stock. One was a wilt disease caused by *Neocosmospora vasinfecta* Smith, which caused losses up to 99 percent of the seedlings in some nurseries.[5] Young seedlings that germinated in May started showing symptoms by the middle of June. The trees wilted suddenly, then shed their leaves and died. The roots of diseased seedlings turned

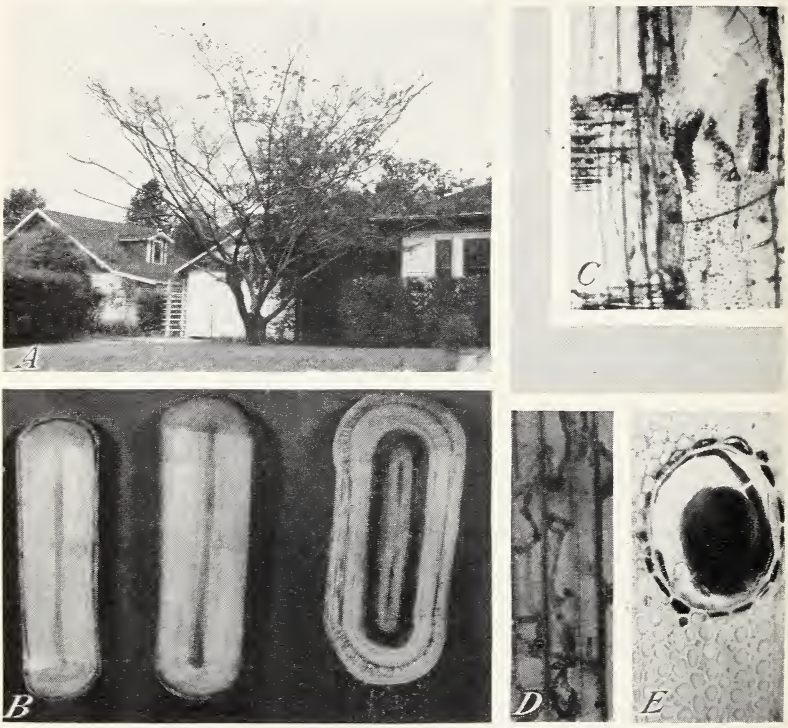


FIGURE 2.—*A*, Mimosa tree succumbing to the disease. This tree had been normal and vigorous up to the summer when this photograph was taken. *B*, Stem sections showing the brown sapwood discoloration typical of diseased trees. *C*, Mycelium in a vessel, and gum deposits in wood parenchyma and ray cells (× 148). *D*, Mycelium in ray cells (× 304). *E*, Gum deposits in wood parenchyma cells and the lumen of a vessel (× 148).

brown. The other disease was a damping-off caused by a species of *Fusarium*.[6]

[5] NISIKADO, Y., and YAMAUTI, K. ON NEOCOSMOSPORA VASINFECTA SMITH, A CAUSAL FUNGUS OF SEEDLING-WILT OF SILK-TREE, ALBIZZIA JULIBRISSIN DURAZ. Ber. Ōhara Inst. Landw. Forsch. 7: 549–556, illus. 1937.

———and YAMAUTI, K. TEMPERATURE RELATIONS TO THE VEGETATIVE AND REPRODUCTIVE GROWTH AND THE PATHOGENICITY OF NEOCOSMOSPORA VASINFECTA SMITH. Ber. Ōhara Inst. Landw. Forsch. 7. 557–572, illus. 1937.

[6] KASAI, M. THE CAUSE OF DEATH OF SILK-TREE SEEDLINGS RAISED IN THE GOVERNMENT RAILWAYS' NURSERIES. Research Off. Japan, Gov. Railways Bull. V. 23, No. 13, 10 pp., illus. 1935. [In Japanese. English summary.]

## ETIOLOGY

### ISOLATIONS

Almost immediately upon death of the foliage and often while the inner bark is still green, fruiting bodies of species of *Fusarium* frequently appear on the bark. Many species have been found, including *Fusarium lateritium* Nees, *F. lateritium* Nees var. *majus* Wr., *F. decemcellulare* Brick, *F. aquaeductuum* (Radlk. and Rabh.) Lagh. var. *medium* Wr., *F. sambucinum* Fckl., *F. stilboides* Wr., and *F. ciliatum* Lk. The most commonly observed were forms of *F. lateritium*. It is significant that none of these fungi belong to the Elegans section, which includes all the species of *Fusarium* known to cause wilts.

Isolations were made from the discolored sapwood of affected trees from eight of the localities previously mentioned (pp. 1 and 2); and Fusaria were obtained, but they were distinct from the species found on the bark. The fungi consistently isolated from the xylem at seven of the localities appeared morphologically alike and belonged in the section Elegans. This form is subsequently referred to in this circular as the Tryon isolate. The isolate from Wisacky, although having many features in common with the Tryon form, differed in some characteristics. In this circular these two types will be referred to as forms of the same fungus. Some of the isolations made at Spartanburg differed in some respects from the common isolate, but they were made too late to be included in the inoculation work. In a few cases *Fusarium solani* Mart. var. *martii* (App. and Wr.) Wr. was also isolated from the discolored wood. This fungus, however, is known commonly to follow primary wilt pathogenes; therefore its occasional presence in the xylem of diseased trees is not surprising.

The consistency with which the Elegans *Fusarium* had been isolated indicted it immediately as the probable cause of the disease. Voronikhin,[7] who investigated the Russian disease, described as the probable cause a *Fusarium* he found fruiting on the bark of killed trees. He does not mention preparing cultures and made no inoculations. He described the fungus in detail and showed that it compared favorably with some other species of *Fusarium*, none of which, however, were wilt fungi. To avoid confusion in nomenclature, he described it as a new species (*Fusarium albizziae*), although it was very similar to certain nonwilt fungi. Since, in the present work, cultures have demonstrated the forms isolated from the wood to be distinct from any of the common bark fungi, and since Voronikhin's fungus apparently did not belong in the Elegans section, it seems likely that he, in describing a fungus that fruited on the bark, was dealing with one of the secondary Fusaria.

### INOCULATIONS

The pathogenicity of the *Fusarium* consistently isolated from discolored wood was tested in several ways. The fungus on agar was inserted in cuts made in the stems of 2- to 3-year-old potted seedlings, but infection did not result. Similar stem inoculations in the field failed. Occasional infection followed root inoculation of seedlings, but most of these inoculations failed.

Since the species of *Fusarium* causing wilt are known to infect from the soil through the roots, trees were grown in inoculated soil.

[7] See footnote 4.

Six flats of sterilized soil were prepared; five of these were heavily inoculated, each with a different fungus, the sixth served as a check. The inocula, which were rice cultures, were worked into the soil. Sterile rice was used for the check. Ten mimosa seedlings, a few weeks old, raised in sterilized soil, were placed in each flat. In addition to these six flats, one flat of unsterilized soil was inoculated with the Wisacky form of the Elegans *Fusarium*. The following tabulation gives the results 7 months after the seedlings were placed in the greenhouse.

| *Fungus used* | *Plants dead at end of test* |
|---|---|
| Xylem fungi: | |
| Elegans *Fusarium* (Wisacky form) | 9 |
| Elegans *Fusarium* (Tryon form) | 8 |
| Elegans *Fusarium* (Wisacky form, unsterilized soil) | 3 |
| *Fusarium solani martii* | 0 |
| Bark fungi: | |
| *Fusarium decemcellulare* | 0 |
| *F. lateritium* | 0 |
| Check | 0 |

The roots of the killed seedlings were washed with sterile water and placed on agar. The fungi used in the inoculations were reisolated in all cases. However, it is to be expected that these fungi would be present on the roots, even if they had not killed the seedlings, since they were present abundantly in the soil. Some of the killed seedlings showed stem and others only root discoloration.

Although these results indicate the suspected fungus to be pathogenic, the killing of such young seedlings may be a quite different process from the killing of grown trees. However, since none of the killed seedlings showed damping-off symptoms and the roots had not been rotted, it seems likely that death was due to vascular invasion.

Seven trees, 1 to 3 inches in diameter at breast height, were inoculated with the suspected Tryon form of the fungus by placing a test tube containing the fungus growing on rice over the end of a small cut root on each tree. Three similar trees were inoculated with the Wisacky form, and four trees were used as checks, the latter having tubes of sterile rice placed over the cut roots. The trees were not disturbed other than to cut off of each the one small root inoculated. At the end of a year four of the seven trees inoculated with the Tryon fungus had wilted, one of the three inoculated with the Wisacky fungus, and one of the four checks. The forms used were reisolated from the infected xylem of the successfully inoculated trees. Since some natural infection was to be expected at Tryon, the one infected check is not surprising. The Tryon form was isolated from this tree.

Successful artificial inoculation with wilt Fusaria has often been difficult and uncertain. However, these inoculation results, together with the fact that the discoloration is heaviest in the roots, suggesting root infection, and the consistent association of a *Fusarium* of the Elegans section strongly indicate that fungus to be the cause of the disease.

## DESCRIPTION OF PATHOGENE

The classification of the species of *Fusarium* has been attended with considerable difficulty and controversy. Classification based solely on either morphologic or physiologic characteristics has not proved completely satisfactory. A lucid account of the difficulties

involved is given by Nelson, Coons, and Cochran.[8] Their problems in classifying the species of *Fusarium* causing the yellows disease of celery were similar to those of the author in attempting to name the forms causing the wilt of *Albizzia*.

The fungi consistently isolated from the wood of wilted trees at all localities from which cultures were prepared, except the Wisacky and some of the Spartanburg cultures, appear identical, with minor variations, and appear not to agree closely in morphology with any of the species of *Fusarium* described by Wollenweber and Reinking.[9] This common isolate, regarded here as the type form of the pathogene, belongs in the subsection Oxysporum of the section Elegans. The outstanding differences between this fungus and the others in that group are: (1) The three-septate macroconidia, which are the most common, are distinctly shorter; (2) spores with more than three septa seldom can be found; and (3) it is pathogenic on *Albizzia*. These characteristics led Sherbakoff (see footnote 2, p. 1) to suggest that this fungus be described as a new species.

For the description that follows, 10 macroconidia of each septation, from each of 9 cultures, were measured. The range covered by the term "mostly" is that delimited by the standard deviation.

***Fusarium perniciosum*** n. sp.

Description refers to the fungus on Difco malt agar. Aerial mycelium light rose purple, submerged mycelium and stroma at first colorless becoming rose purple and finally magenta or darker. Microconidia abundant, typically ovoid, one-celled, produced mostly on aerial mycelium, usually appearing in false heads, in mass white to buff pink. Macroconidia usually produced on sporodochia, sometimes on aerial mycelium, typically three-septate, curved, gradually attenuated toward both ends, usually with distinct foot cells. 0-septate mean $8\mu \times 3.1\mu$, mostly $5\mu$–$11\mu \times 2.5\mu$–$3.7\mu$; one-septate, $17\mu \times 3.8\mu$, mostly $14\mu$–$19\mu \times 3.1\mu$–$4.5\mu$; two-septate, $21\mu \times 4.1\mu$, mostly $18\mu$–$23\mu \times 3.4\mu$–$4.8\mu$; three-septate, $28\mu \times 4.4\mu$, mostly $23\mu$–$32\mu \times 3.9\mu$–$4.9\mu$. Spores with more than three septa usually absent. Chlamydospores terminal, intercalary, intraconidial, or attached to conidia, globose to pyriform, mean diameter $7\mu$. No sclerotia, but masses of plectenchymatous tissue form early. These are soft, brown inside, with a surrounding sheath of white mycelium. Observed variations from normal include cultures with stroma and aerial mycelium flesh pink, cultures with no aerial mycelium and macroconidia in pionnotes, cultures without macroconidia, and cultures without plectenchymatous bodies. Parasitic on *Albizzia julibrissin* Duraz.; probable cause of vascular wilt of this tree in the southeastern United States.

Mycelio pallide roseopurpureo usque magenteo; microconidiis copiosis, typice ovoideis, unicellularibus, in massis albis vel alutaceo roseis; macroconidiis in sporodochiis, interdum in mycelio aerio productis, 1–3 septatis, curvatis, utrinque attenuatis, $5\mu$–$32\mu \times 2.5\mu$–$4.9\mu$, typice 3-septatis, $23\mu$–$32\mu \times 3.9\mu$–$4.9\mu$; chlamydosporis terminalibus, intercalaribus, intraconidiis vel conidiis affixis, globosis vel pyriformibus, $7\mu$ diam.

Macroconidia of the Wisacky form were obtained in only two cultures. Three-, four-, and five-septate spores were common, with the average dimensions of the three-septate $39.7\mu \times 4.6\mu$. The occasional production of small sclerotia, together with the differences in spore characteristics, separate this form from the more common one. The two forms are similar in color and in the formation of plechtenchymatous masses on malt agar. Further study of the Wisacky form is needed before it can be adequately described.

The isolations from Spartanburg yielded typical *Fusarium perniciosum* and a *Fusarium* that produced dark sclerotia and darkened the

[8] Nelson, R., Coons, G. H., and Cochran, L. C. The fusarium yellows disease of celery, apium graveolens l. var. dulce dc. Mich. Agr. Expt. Sta. Tech. Bull. 155, 74 pp., illus. 1937.

[9] Wollenweber, H. W., and Reinking, O. A. Die fusarien, ihre beschreibung, schadwirkung und bekämpfung. 355 pp., illus. Berlin. 1935.

surface of the agar. The two fungi were alike in spore septation, size, and shape, and were similar in many other respects. The answer to whether the Wisacky and Spartanburg forms are morphologic variants of *F. perniciosum* or distinct fungi awaits further work.

### COMPARISON OF FUSARIUM PERNICIOSUM WITH RELATED FUNGI PARASITIC ON ALBIZZIA JULIBRISSIN

The major differences between *Fusarium perniciosum* and the fungi reported as causing seedling diseases of *Albizzia julibrissin* in Japan [10] are given in table 1.

The differences among the American and Japanese fungi are sufficiently marked so that these organisms are regarded as distinct. Since Voronikhin [11] did not prepare tissue cultures from infected xylem it would mean little to compare the American fungus with the Russian.

TABLE 1.—*Comparison of Fusarium perniciosum with related fungi parasitic on Albizzia julibrissin*

| Fungi | Type of disease | Conidia | Sporodochia | Perithecia |
|---|---|---|---|---|
| *Fusarium perniciosum.* | Vascular wilt of trees of all sizes. | *Cephalosporium* and *Fusarium* types common, the latter 1- to 3-septate. | Usually produced. | None. |
| *Neocosmospora vasinfecta.* | Reported only as 1-year seedling wilt. | Mostly *Cephalosporium*, some *Fusarium* type, the latter 1- to 3-septate. | None reported | Produced readily. |
| Kasai's *Fusarium* | Damping-off | *Cephalosporium* and *Fusarium* types, the latter 1- to 5-septate, shape distinct from others. | do | None. |

### EPIDEMIOLOGY

The disease has been found in mimosa trees growing on soils ranging from clay in the Piedmont to almost pure sand in the Coastal Plain, from elevations near sea level to those of 2,200 feet at Biltmore, and on soils with pH values of 4.5 to 7.8. The known infection areas form no definite geographical pattern and so far appear to have no marked common factors that would account for the presence of the disease at those particular places.

About the middle of August 1938 a survey of the mimosa trees more than about 2.5 inches in diameter at breast height in Tryon showed 38 percent of the 280 trees examined to be dead or dying. Since at least 300 dead trees have been removed in the last few years, the mortality of the street trees of Tryon due to the wilt has been about 70 percent.

The epidemic nature of the disease in the Soviet Union and in the United States suggests that it is not native where found in either country. *Albizzia julibrissin* apparently has been grown in the affected areas in the Soviet Union for many years and at Tryon since some time during the nineteenth century at the latest. Owners of mimosa trees and residents in towns in the South where the tree is common and where the disease has now appeared were questioned as to whether they knew of any dying of this tree in the past. In no case was the tree known to have had a serious enemy. The sudden devastating occurrence of the disease could hardly be consistent

[10] See footnotes 5 and 6.
[11] See footnote 4.

with the presence of the causal organism for a great many years. Since there seems to be no diminution in the severity of attack so far, the disease must be considered a distinct threat to the existence of the mimosa tree in the United States.

## CONTROL

As it is probable that this disease is largely or wholly soil-borne, it would be most difficult to check if it once became established in a given locality. However, certain preventive measures would likely retard its rate of spread from place to place. The movement of soil, directly or attached to the roots of plants, from areas of known infection should be discouraged. Not only diseased mimosa trees but any plants with infested soil adhering to them could probably establish the disease in new localities. Planting stock should be selected from areas definitely known to be free from the disease.

Where the disease is established, no practical way is known to prevent its spread from tree to tree. The growth of the fungus through the soil, the movement of infested soil during rains, and the transportation of the soil on automobile tires and by other means will eventually probably cause most of the soil capable of harboring the parasite, in a given locality, to become infested. Where the disease has appeared, it would be best to replace the mimosa with other species, since that tree may be killed out at those places.

Although it has not yet been definitely established whether infection can take place through wounds on the trunk or branches until definite information is obtained to the contrary, pruning or other wounds should be disinfected immediately and covered with coal tar, asphaltum, or some other serviceable coating. Before disinfecting, the cambium should be protected with shellac. If any pruning or other work with tools is done on trees suspected of having the disease, the tools should be disinfected with bichloride of mercury or some other effective disinfectant before using them on healthy mimosas.

## SUMMARY

A highly destructive vascular wilt disease of the so-called mimosa tree (*Albizzia julibrissin* Duraz.) has appeared within the past few years in the Southeast.

The principal symptoms are wilting, followed by death of the foliage, and a brown ring of discolored sapwood, usually in the current annual ring of the stem and branches.

A disease similar in its destructive habit and with symptoms identical with those of the disease in the Southeast has been described from the southern Soviet Union.

What appear to be two forms of a member of the section Elegans of the genus *Fusarium* have been isolated from the discolored sapwood of diseased trees. Successful inoculations have been made with these forms. The form most commonly isolated is described as a new species, *Fusarium perniciosum*.

Several species of *Fusarium* were often found fruiting on the bark of trees dying or dead from the wilt disease, but all were distinct from the vascular fungi, and none of them belonged in the section Elegans, which includes all known wilt-causing species of *Fusarium*.

The disease has been found in trees growing on soils ranging from sand to clay, from acid to alkaline, and in elevation from near sea level to 2,200 feet. The localities where the disease has been found appear to form no definite geographical pattern.

Several thousand trees have been killed in the 8 years the disease has been known in the Southeast. Because the disease is a rapid killer and because it appears to have spread since about 1930 from Tryon, N. C., where it was first discovered, to a large number of other widely scattered localities, it must be considered a real threat to the mimosa tree in the United States.

The movement of soil, directly or attached to the roots of plants, from areas of known infection to areas where the mimosa tree is valued should be discouraged. Mimosa planting stock should be selected from areas definitely known to be free from the disease.

Where the disease has appeared in a locality, no practical way is known to check its spread from tree to tree. It would be best in such cases to replace the mimosa with other species, since it is likely that species can no longer be successfully grown there.

As a precaution, wounds should be disinfected and covered with some serviceable coating. If pruning or other work with tools is done on trees suspected of having the disease, the tools should be disinfected before they are used on healthy mimosas.

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